AETIOLOGICAL PROFILE AND CLINICO-ECHOCARDIOGRAPHIC FEATURES OF CONGESTIVE HEART FAILURE

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GOVT. STANLEY MEDICAL COLLEGE & HOSPITAL
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CERTIFICATE

This is to certify that the dissertation titled **"AETIOLOGICAL PROFILE AND CLINICO ECHOCARDIOGRAPHIC FEATURES OF CONGESTIVE HEART FAILURE"** is the bona fide original work

of **Dr. K. VASANTH** in partial fulfillment of the requirements for **M.D.**

Branch – I (General Medicine) Examination of the Tamil Nadu Dr. M.G.R Medical University to be held in MARCH 2009. The period of study was from August 2007 to August 2008.

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DECLARATION

I, Dr. K. VASANTH hereby solemnly declare that the dissertation titled **"AETIOLOGICAL PROFILE AND CLINICO ECHOCARDIOGRAPHIC FEATURES OF CONGESTIVE HEART FAILURE"** was done by me at Govt. Stanley Medical College and Hospital from August 2007 to August 2008 under the supervision and guidance of my Unit Chief and **Head of Department of Medicine Prof.V.RUCKMANI, M.D.,**

This dissertation is submitted to Tamil Nadu DR. M.G.R Medical University, towards partial fulfillment of requirement for the award of **M.D. Degree (Branch - I) in General Medicine.**

Place: Chennai.
Date:

(DR. K. VASANTH)

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INDEX

INTRODUCTION

Heart failure is the end stage of all diseases of the heart and is a major cause of morbidity and mortality. Since 1970s the treatment of CHF`has been transformed, resulting in major benefit to patients. This advance has been the consequence of better understanding of the pathophysiology, investigations, the introduction of newer drugs and cardiac transplantation. The traditional treatment of heart failure with digoxin and diuretics has been replaced by diuretics, ACE inhibitors and drugs directed against the origins of heart failure such as aspirin and lipid lowering drugs. Newer objectives are optimization of the quality of life, avoidance of hospital admissions prevention of progression of damage to the myocardium and prolongation of life.

So it becomes important to conduct clinical and Para clinical studies to know about the status, precipitating factors and complications of the disease. Only with a reliable study, changes in the modality of approach in controlling, diagnosing and treating the disease can be done. Here, an attempt has been made to study on selected aspects of congestive heart failure.

REVIEW OF LITERATURE

History:

Heart failure, angina and the pulse were known in the ancient Egyptian and early Greek civilizations. (Dallas 1993, Horine 1941). Hippocrates described cardiac cachexia, most vividly reports of the benefits of foxglove exists in Roman literature. (Moore, 1985) Hering used nitrate in 1853 to treat heart failure, the first use of vasodilator. Bruton later in 1867 described the use of amyl nitrate to treat angina. ACE inhibitors were shown to be of benefit in terms of mortality in patients with heart failure for the first time in 1987. The large trail of digoxin, showing no effect on overall mortality, was reported in 1996.

Definitions of heart failure

1. A pathophysiological state in which an abnormality of cardiac function is responsible for the failure of the heart to pump blood at a rate commensurate with the requirements of the metabolizing tissues (Braunwald 1994).
2. Congestive heart failure represents a complex clinical syndrome, characterized by abnormalities of left ventricular function and neurohormonal regulation, which are accompanied by effort intolerance, fluid retention and reduced longevity.(Packer 1988).
3. Symptoms of heart failure, objective evidence of cardiac dysfunction and response to treatment directed towards the heart failure. (Task force of the European society of cardiology 1995)

EPIDEMIOLOGY:

Heart failure is a relatively common disorder. It is estimated that 4.6 million persons in the United States are being treated for heart failure, with 550,000 new cases diagnosed each year. (Dallas 1999, Massie - 1977)

The prevalence of heart failure increases dramatically with age, occurring in 1 to 2 percent of persons aged 50 to 59 and up to 10 percent of individuals older than the age of 75. (Ho et al 1993)

Approximately 80 percent of all heart failure admissions occur in patients older than 65, as a result, heart failure is the leading discharge diagnosis in persons 65 years or older in the United States (Rich 1999).

Despite a steady decline in the incidence of coronary artery disease and stroke, both the incidence and prevalence of heart failure continue to rise.

Between 1985 and 1995 the number of heart failure hospitalizations increased by 5 percent and 8, 70,000 hospital discharges for heart failure occurred in 1996. (Haldeman – 1999)

In the United States approximately 45,000 deaths each year are primarily caused by heart failure and heart failure is listed as a contributing cause in 260,000 deaths (Dallas 1999).

In smaller mid western areas, recent assessment has suggested a prevalence as high as 6% of population. The same prevalence of 6-7% also has been observed in Urban population. (Senni et al - 1999)

The overall prevalence of heart failure is 3-20 per 1000 population although this exceeds 100 per 1000 in those aged 65 yrs and over. The annual incidence of heart failure is 1-5 per 1000 and the relative incidence doubles for each decade of life after the age of 45 yrs. (Davis Hobbs, Lip – 2003). The overall incidence is likely to increase in the future because of both an aging population and therapeutic advances in the management of acute myocardial infarction leading to improved survival in patients with impaired cardiac function. (Cowie et al - 1999).

Three major factors such as age, race and gender influence the prevalence and outcome in patients with heart failure.

Age:

The most important factor is age. The prevalence of CHF is less than 1% in patients < 50 yrs of age regardless of gender. At older than 50 years of age however, the prevalence increases to approximately 5% for patients between 50 and 70 years of age, nearly 10% for all patients over 70 yrs of age and perhaps as high as 15% for patients over the age of 80.

The impact of age alone leads to a significant continued increase in the prevalence of heart failure as recent statistics have suggested that there will be near doubling of patients over 65 years of age by the year 2030. (Dallas 2001).

Race:

The second major influencing factor is race. There has been a higher prevalence of heart failure in blacks compares to whites.

More importantly black patients develop heart failure at a younger age than white patients. (Bourassa et al 1993).

Gender:

The third major factor influencing the heart failure is gender. The national heart and nutrition education survey (NHANES) from 1988-1994 estimated that there is an increased prevalence of congestive heart failure in men aged 70 years and younger. In contrast, women aged older than 70 years have a higher prevalence. This difference may be in part owing to the increased average life expectancy in women (Anderson 2001).

The highest prevalence of CHF is in black men followed by black women.

Data derived from the National centre of Health Statistics report that total life expectancy for the United States Population is 76.7 years, Life expectancy is 73.8 years for men and 79.5 years for Women (2000 estimate).

While the overall incidence of congestive heart failure is probably equal between the two genders there are several unique features that may influence the prevalence of congestive heart failure in women, including a higher average ejection fraction for an equivalent amount of symptoms than men at all ages. This may reflect a higher prevalence of primary diastolic rather than systolic dysfunction. (Gheorghide, Bonow

1998)

The Framingham data shows an age adjusted annual incidence of heart failure of 0.14% in women and 0.23% in men. Survival in women is generally better than in men.

AETIOLOGY:

The relative importance of aetiological factors in heart failure is dependent on the nature of the population being studied, as coronary artery disease and hypertension are common causes of heart failure in western countries, whereas valvular heart diseases and nutritional cardiac diseases are more common in the developing world. (Zannad - 1999)

The common causes of heart failure include:

1. coronary artery disease, Ischemia
2. hypertension
3. cardiomyopathies
4. valvular heart disease and congenital heart diseases
5. arrhythmias
6. alcohol and drugs
    - alcohol
    - cardiac depressant drugs (beta blockers and calcium channel blockers)

7. high output failure
    - anemia, thyrotoxicosis, beriberi, etc
8. pericardial diseases
9. primary right heart failure

    -Pulmonary hypertension eg. pulmonary embolism, corpulmonale

    -tricuspid incompetence

Coronary artery disease and its risk factors:

Coronary artery disease is the commonest cause of heart failure. In the studies of left ventricular dysfunction (SOL VD) Coronary artery disease accounted for about 75% of the cases of chronic heart failure.

(Khadra Saleem, Rand 1998)

Coronary artery disease and hypertension (either alone or in the combination) were implicated as the cause in over 70% of cases of heart failure in the fragmentation study. (Ho – 1993)

Coronary risk factors, such as smoking and diabetes are also risk markers for the development of heart failure. Smoking is an independent and strong risk factor for the development of heart failure in men, although the findings in women are less consistent.

In Framingham heart study, diabetes and left ventricular hypertrophy were the most significant risk markers for the development of heart failure. Body weight and high ratio of total cholesterol concentration to high density lipoprotein

cholesterol concentration are also independent risk factors for heart failure. Clearly these risk factors may increase the risk of heart failure through their effects on coronary artery disease, although diabetes alone may induce important structural and functional changes in the myocardium, which further increase the risk of heart failure.

Hypertension:

Hypertension has been associated with an increased risk of heart failure in several epidemiological studies. In the Framingham heart study, hypertension was reported as the cause of heart failure either alone or in association with other factors, in over 70% of cases on the basis of non invasive assessment. (Ho - 1993). However hypertension is probably a more common cause of heart failure in selected patient groups, including females and black populations. Hypertension predisposes to the development of heart failure via a number of pathological mechanisms, including left ventricular hypertrophy.

Left ventricular hypertrophy is associated with left ventricular systolic and diastolic dysfunction and an increased risk of myocardial infarction and it predisposes to both atrial and ventricular arrhythmias. Electrocardiographic left ventricular hypertrophy is strongly correlated with the development of heart failure, as it associated with a 14 fold increase in the risk of heart failure in those aged 65 years or under.

Valvular heart disease:

Rheumatic heart disease may have declined in certain parts of the world, but it still remains an important cause of heart failure in India and

other developing nations. In the Framingham heart study, rheumatic heart disease accounted for heart failure in 2% of men and 3% of women,
although the overall incidence of valvular heart disease has been steadily
decreasing in the Framingham cohort over the past 30 years (HO - 1993).

MR and AS are the most common causes of heart failure, secondary to valvular disease. MR and AR lead to volume overload, in contrast with AS which leads to pressure overload. The progression of heart failure in patients with valvular heart disease is dependent on the nature and extent of the valvular disease. In aortic stenosis heart failure develops at a relatively late stage and without, valve replacement, it is associated with a poor prognosis. In contrast, patients with chronic mitral or aortic regurgitation generally decline in a slower and more progressive manner. (Teerlink, et al 1991).

Cardiomyopathies:

Cardiomyopathies are defined as the disease of the heart muscle that are not secondary to coronary heart disease, hypertension or others. As primary disease of heart muscle, cardiomyopathies are less common causes of heart failure, but awareness of their existence is necessary to make a diagnosis. Cardiomyopathies are separated into four functional categories dilated, hypertrophic, restrictive and obliterative. These groups can include rare specific heart muscle diseases such as hemochromatosis in which cardiac involvement occurs as part of a systemic disorder. Dilated cardiomyopathy is a more common cause of heart failure than hypertrophic and restrictive Cardiomyopathies, obliterative cardiomypathy is essentially limited to

developing countries. (Oakley 1997).

Arrhythmias:

Cardiac arrhythmias are more common in patients with heart failure and associated structural heart diseases, including hypertensive patients with left ventricular hypertrophy.

In the Hillingdon heart failure study 30% of patients presented for the first time with heart failure had atrial fibrillation and over 60% of patients admitted urgently with atrial fibrillation to a Glasgow hospital had echocardiographic evidence of impaired left ventricular function (Stevenson, 1995).

Alcohol and drugs:

Alcohol has a direct toxic effect on the heart which may lead to acute heart failure or heart failure as a result of arrhythmias, commonly atrial fibrillation. Excessive chronic alcohol consumption also leads to dilated cardiomyopathy.

Alcohol is the identifiable cause of heart failure in 2-3% of cases.
Chemotherapeutic agents (doxorubicin) and antiviral drugs (zidovudine)
have been implicated in heart failure, through direct toxic effects on the
myocardium. (Maki et al - 1998)

Endocrine causes:

High output heart failure is most often seen in patients with anaemia and thyrotoxicosis. Myxedema may present with heart failure as a result of myocardial involvement of secondary to pericardial effusion.

Corpulmonale:

Corpulmonale is defined as enlargement of the right ventricle secondary to abnormalities of the lungs, thorax, pulmonary ventilation or circulation. It sometimes leads to right ventricular failure, with an elevation of trasnsmural right ventricular end diastolic pressure. (Rich et al 2005).

Pericardial Diseases:

Pericardial diseases like tuberculosis, CRP and malignant involvement may also cause congestive heart failure.

Nutritional Causes:

Vitamin deficiency like wet beriberi, anaemia and deficiency of hematopoetic factors leading to heart failure, continue to produce a problem especially in developing countries like India.

PATHOPHYSIOLOGY:

Heart failure is the multisystem disorder which is characterized by the abnormalities of cardiac, skeletal muscle and renal function with stimulation of the sympathetic nervous system and a complex pattern of neurohormonal changes.

Myocardial Systolic Dysfunction:

The primary abnormality in non valvular HF is an impairment in the left ventricular (LV) function leading to fall in cardiac output. This fall in cardiac output leads to activation of several neurohormonal compensatory mechanisms aimed at improving the mechanical environment of heart. Activation of sympathetic system tries to maintain cardiac output with increase in heart

rate, increases myocardial contractility and peripheral vasoconstriction. Activation of Renin-Angiotensin-Aldosterone system (RAAS) results in vasoconstriction and increase in blood volume with salt and water retention. Concentration of vasopressin and natriuretic peptides increase. Furthermore there may be progressive cardiac dilatation or alterations in cardiac structure or both (Borgeon, Burnett, 1997).

Renin – Angiotensin – Aldesterone system (RAAS)

Simulation of RAAS leads to increased concentration of Renin, Angiotensin II (AT II) and Aldosterone. AT II is a potent vasoconstrictor of renal and systemic circulation where it stimulates release of Nor Adrenaline from sympathetic nerve terminals, which inhibits vagal tone and promotes release of Aldosterone. This leads to sodium and water retention, in addition, AT – II has an important effects on cardiac myocytes and may contribute to endothelial dysfunction. (Francis et al, 1990)

Sympathetic Nervous System:

Sympathetic nervous system is activated in HF via low and high pressure baroreceptors as an early compensatory mechanism which provides inotropic support and maintain cardiac output. In long term, the ability of the myocardium to respond to chronic high concentration of catecholamines is activated by down regulation of ß-receptors. This may be associated with baroreceptor dysfunction and further increase in sympathetic activity (Schoffer et al, 1987).

Natriuretic Peptides:

There are three natriuretic peptides of similar structure and these exert a wide range of effects on heart, kidneys and cardio vascular system.

Atrial natriuretic peptide (ANP) is released from atria in response to stretch, leading to natriuresis and vasodilatation. In humans, Brain natriuretic peptide (BNP) is also released from heart and its actions are similar to those of ANP. C- type natriuretic peptide is limited to vascular endothelium and CNS and has only limited effects on natriuresis and vasodilatation. (Van Chang et al, 2001 and Moe et al, 1993)

Vasopressin:

Vasopressin concentration is also increased in severe chronic HF. High concentration of the hormone are particularly common in patients receiving diuretic treatment and this may contribute to development of hyponatremia (Francis et al, 1990 and Goldsmith, 1986).

Endothelin:

Secreted by vascular endothelial cells, is a potent vasoconstrictor on renal vasculature. Its concentration is also correlated with indices of severity such as pulmonary capillary wedge pressure (PCWP) and need for hospitalisation and death (Tsutamatoet al, 1995).

Patterns of Neurohormonal activation and prognosis

Asymptomatic left ventricular dysfunction:

Plasma noradrenaline concentration increases early in development of left ventricular dysfunction and plasma renin activity usually increases in patients receiving diuretic treatment. Nor adrenaline concentration in patients with asymptomatic LV dysfunction is a strong and independent predictor of development of symptomatic HF and long term morality.

In severe untreated chronic HF, concentrations of renin, AT – II, Aldosterone, nor adrenaline and ANP are all increased. Plasma levels correlate with both the severity of HF and the long term prognosis. Patients with chronic HF and increased plasma nor adrenaline concentration do also have a worse prognosis (Esler, 1997).

Diastolic dysfunction:

Diastolic dysfunction refers to clinical syndrome of HF with preserved Left Ventricle Ejection Fraction (EF – 0.4 or more) in the absence of major valvular disease. In diastolic HF, LV cavity is stiff due to increased LV mass. It relaxes slowly in early diastole and offers greater resistance to filling in late diastole so that diastolic pressure is increased. The low cardiac output manifests as fatigue while the increase in end diastolic pressure is transmitted backwards through valve less pulmonary veins to pulmonary capillaries resulting in exertional dyspnea (Vasa Levy, 2000).

Mechanisms contributing to abnormal LV diastolic properties include stiff arteries, hypertension, ischemia, Diabetes and intrinsic

myocardial changes with or without associated hypertrophy (Kitzman et al, 2002). The prognosis of diastolic HF is generally better than that of systolic Heart failure.

Diastolic Heart failure is common in clinical practice. The Diagnosis of diastolic HF may be considered in patients with HF who have normal LV EF (0.4 or more) (Ibrahim, 2003).

Myocardial Dysfunction due to Remodeling, Hibernation and Stunning:

After extensive myocardial infarction, cardiac contractility is frequently impaired and neurohormonal activation leads to regional eccentric and concentric hypertrophy of Non-infarcted segment with expansion of infarct zone. This is known as Ventricular Remodeling. Particular risk factors for this development of progressive ventricular dilatation after an MI include large infarcts, anterior infarctions, occlusion of artery related to infarction and hypertension.

Myocardial dysfunction may also occur in response to stunning which describes delayed recovery of myocardial function despite restoration of coronary blood flow in the absence of irreversible damage. This is in contrast to hibernating myocardium which describes persistent myocardial dysfunction due to reduced perfusion although cardiac myocytes remain viable and myocardial contraction may improve with revascularization (Rahimtoola, 1989).

TYPES OF HEART FAILURE

Forward Failure:

It is defined as the inability of the heart to maintain effective stroke volume to meet the metabolic demands of the body.

Backward Failure:

Small transient inequality between the two ventricles resulting in acute pulmonary edema.

Right sided heart failure:

Due to stagnation of blood in the right side of the heart and right heart failure signs develop.

Left sided heart failure:

Due to poor LV contractile function in aortic stenosis or massive MI where the signs of left sided heart failure develops.

Acute heart failure Vs chronic heart failure:

The clinical manifestations of HF depends mainly on the rate at which the syndrome develops and specifically whether sufficient time has elapsed for compensatory mechanisms to become operative and for fluid to accumulate in the interstitial space.

Low output Vs high output failure:

Heart failure in low cardiac output at rest or in milder cases during exertion characterizes most forms of cardiovascular diseases.

High output heart failure occurs in variety of high output states like thyrotoxicosis, Paget's disease, AV fistulas anaemia and beriberi. (Stevenson 1989).

Systolic Vs diastolic HF:

Implicit in the physiological definition of heart failure (inability to pump an adequate volume of blood and or to do so only from an abnormally elevated filling pressure) is that heart failure can be caused by an abnormality in systolic function leading to a defect in expulsion of blood leading to systolic heart failure or by an abnormality in diastolic function leading to a defect in ventricular filling (diastolic failure). This may be due to slowed or incomplete ventricular relaxation, which may be transient as occurs in ischaemia, or sustained, as occurs in concentric myocardial hypertrophy or restrictive cardiomyopathy secondary to infiltrative conditions like amyloidosis.

The principal clinical manifestations of systolic failure result from an inadequate cardiac output and secondary salt and water retention – forward heart failure (Gaasch 1994).

Whereas the major consequence of diastolic HF is related to the elevation of ventricular filling pressure upstream to the ventricular cavity, causing pulmonary and systemic congestion (backward failure). (Vasan

1995)

Clinical features and complications:

Patients with HF present with a variety of symptoms, most of which are nonspecific. The common symptoms of congestive HF include fatigue, dyspnoea, pedal edema and exercise intolerance or symptoms that relate to the underlying cause.

Symptoms and signs in heart failure:

Symptoms

Dyspnoea

Orthopnoea

Palpitation

Chest pain

Reduced exercise tolerance, lethargy, fatigue

Nocturnal cough

Wheeze

Ankle swelling

Anorexia

Signs

Cachexia and muscular wasting

Tachycardia

Pulsus alternans

Increased JVP

Displaced apex beat

RV heave

Crepitations or wheeze

3rd heart sound

Oedema

Hepatomegaly, ascites

Symptoms:

Dyspnoea:

Exertional breathlessness is the frequent presenting symptom in heart failure, although it is a common symptom in patients with pulmonary disease. Dyspnoea is therefore moderately sensitive but poorly specific for the presence of HF.

NYHA Classification of dyspnea:

Class I – no limitation of physical activity. ordinary physical activity does not cause undue symptoms.

Class II – slight limitation of physical activity. ordinary physical activity causes undue fatigue, dyspnea or palpitation.

Class III – moderate limitation of physical activity. less than ordinary activity causes symptoms.

Class IV – Severe restriction of physical activity. unable to perform to any activity without symptoms.

Orthopnea:

Orthopnea is a more specific symptom. PND results from increased LV filling pressure and therefore has a greater sensitivity and predictive value (Maning 1995).

Fatigue and Lethargy

Fatigue and Lethargy in CHF are due to impaired muscle blood flow and poor tissue perfusion.

Oedema

Swelling of ankles and feet is another common presenting feature. Heart failure may manifest as oedema, right hypochondrial pain (liver congestion) and loss of appetite (due to bowel congestion). An increase in weight may be associated with fluid retention although cardiac cachexia and

weight loss are important markers of disease severity. (Milne 1985)

Physical Signs

Physical examination has serious limitations as many patients particularly those with less severe heart failure, have few abnormal signs. In addition some physical signs are difficult to interpret, and if present, may occasionally be related to diseases other than HF.

Oedema and tachycardia, for example are too intensive to have any useful predictive value and although pulmonary crepitations may have a high diagnostic specificity. Increased JVP has a high specificity in diagnosing HF in patients who are known to have cardiac disease.

Displaced apex beat in patients with myocardial infraction and 3rd heart sound have a relatively high specificity.

Framingham Criteria for Diagnosis of Congestive Heart Failure:

Major Criteria

1) Paroxysmal nocturnal dyspnea
2) Neck vein distension
3) Rales

4) Cardiomegaly

5) Acute pulmonary edema

6) S3 gallop

7) Increased Venous Pressure (>16cm H_2O)

8) Positive Hepatojugular reflux

Minor Criteria:

1) Extremity edema

2) Night Cough

3) Dyspnea on exertion

4) Hepatomegaly

5) Pleural effusion

6) Vital capacity reduced by one-third from normal

7) Tachycardia (≥ 120 bpm)

Major or Minor:

Weight loss ≥ 4.5 kg over 5 days of treatment

To establish a clinical diagnosis of congestive heart failure by these criteria at least one major and two minor criteria are required.

AIM and OBJECTIVES

1) To study about the etiological profile of congestive heart failure.

2) To study about the clinical features of congestive heart failure.

3) To study about the Echocardiographic features of congestive heart failure

MATERIALS and METHODS

Place : Department of Medicine

Design : Observational Study

Period : August 2007 to August 2008

Sample Size : 100 Patients

Collaborating : Cardiology

Departments

Inclusion Criteria

1) All patients above 12 years of age with clinical evidence of congestive heart failure.
2) Patients of Both sexes.

Exclusion Criteria

1) Patients less than 12 years of age
2) Hemodynamically unstable patients
3) Pregnant women
4) Patients with Cor Pulmonale

METHODS:

1. Thorough history.

2. Complete physical examination.

3. Chest X-ray.

4. ECG.

5. Complete Blood Count

6. Renal Function Tests.

7. Echo cardiography

Definitions used for the study

1) **Dyspnea:**

   Subjective awareness of the sensation of breathing

2) **Orthopnea:**

   Dyspnea on assuming recumbency.

3) **Angina:**

   Retrosternal chest discomfort due to myocardial ischemia.

4) **Palpitation:**

   Abnormal and uncomfortable awareness of one own heart beat.

Diabetes:

American diabetes association criteria for the diagnosis of diabetes mellitus.

1. Symptoms of diabetes plus blood glucose concentration ≥ 200 mg/dl or
2. Fasting plasma glucose ≥ 126 mg/dl or
3. Two hour plasma glucose ≥ 200 mg/dl during an oral glucose tolerance test.

Hypertension:

Clinical blood pressure of > 140/90 mm Hg confirmed on two separate occasions. (JNC > criteria)

Ischemic Heart Disease:

IHD and CAD is typically defined as a > 50% stenosis of any epicardial coronary artery.

Manifestations of CAD include stable angina, acute coronary Syndrome (ACS), congestive heart failure, sudden cardiac death and silent ischemia.

MI – Regional wall Motion abnormalities in Transthoracic Echo

cardiography

Hypokinesia

Akinesia

Dyskinesia

Rheumatic Heart Disease:

Evidence of valvular involvement in the form of thickening, fibrosis or calcification.

Ejection Fraction:

Depressed ejection fraction < 40%

Normal ejection fraction > 50%

Dilated Cardiomyopathy:

Demonstration of global hypokinesia of left ventricle without regional wall motion abnormalities.

OBSERVATIONS & DATA ANALYSIS

Total Number of Patients – 100

Male - 67

Female - 33

Mean Age of Patients = 50 yrs

TABLE - 1

AGE(Years)	MALE	FEMALE	TOTAL (n=100)
12 - 20	4	4	8
21 - 30	5	2	7
31 - 40	5	4	9
41 - 50	12	9	21
51 - 60	21	9	30
61 - 70	15	3	18
> 70	6	1	7

AGE & SEX DISTRIBUTION

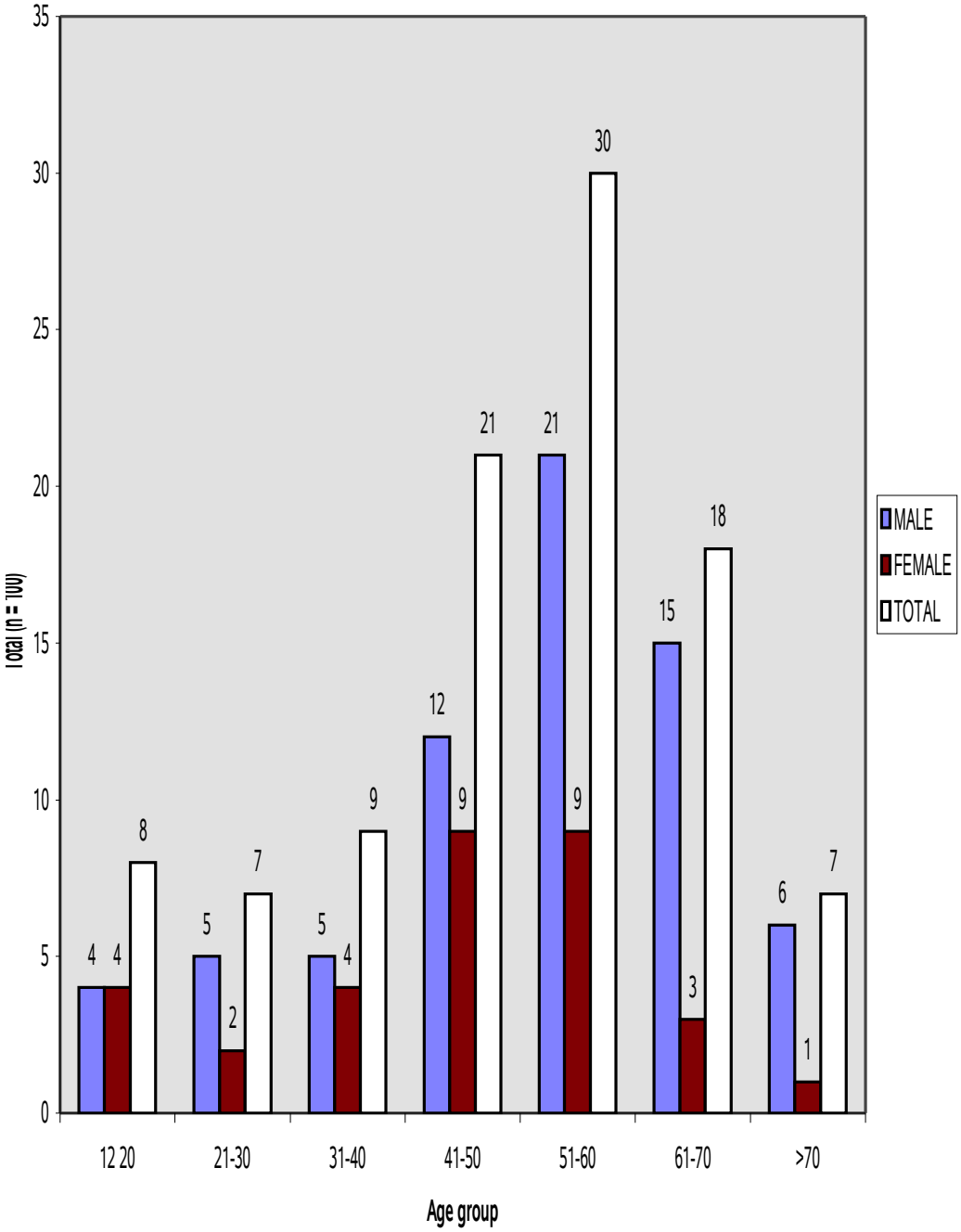


TABLE - 2

PRESENTING FEATURES

Dyspnea class	Male	Female	Total(n=100)
Class I	0	0	0
Class II	2	0	2
Class III	28	11	39
Class IV	37	22	59

Dyspnea is the most common presenting symptom. 100% of patients had dyspnea. Majority (59%) of patients were in Class IV failure.

PRESENTING FEATURES

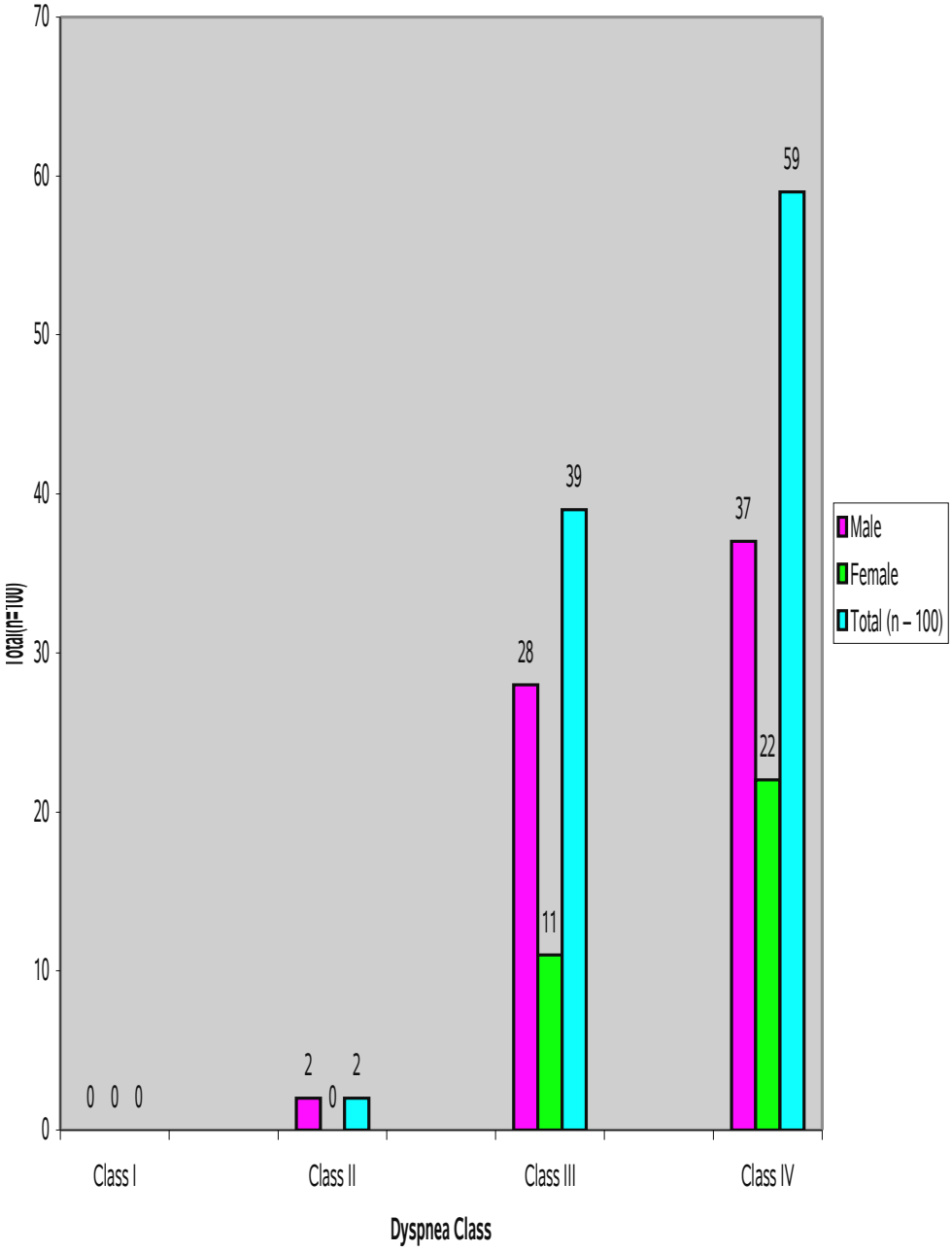


TABLE - 3

SYMPTOM COMPLEX

Symptoms	Males	Females	Total(n=100)
Orthopnea	63(94%)	32(97%)	95
PND	25(37%)	13(39%)	38
Fatigue	59(88%)	31(94%)	90
Pedal Edema	54(80%)	24(72%)	78
Angina	20(30%)	9(27%)	29
Palpitation	22(33%)	21(63%)	43
Night Cough	45(67%)	17(51%)	62

Next to dyspnea, Orthopnea is the most common symptom,

followed by fatiguability and pedal edema. Significant percentage of females had palpitation compared to males.

SYMPTOM COMPLEX

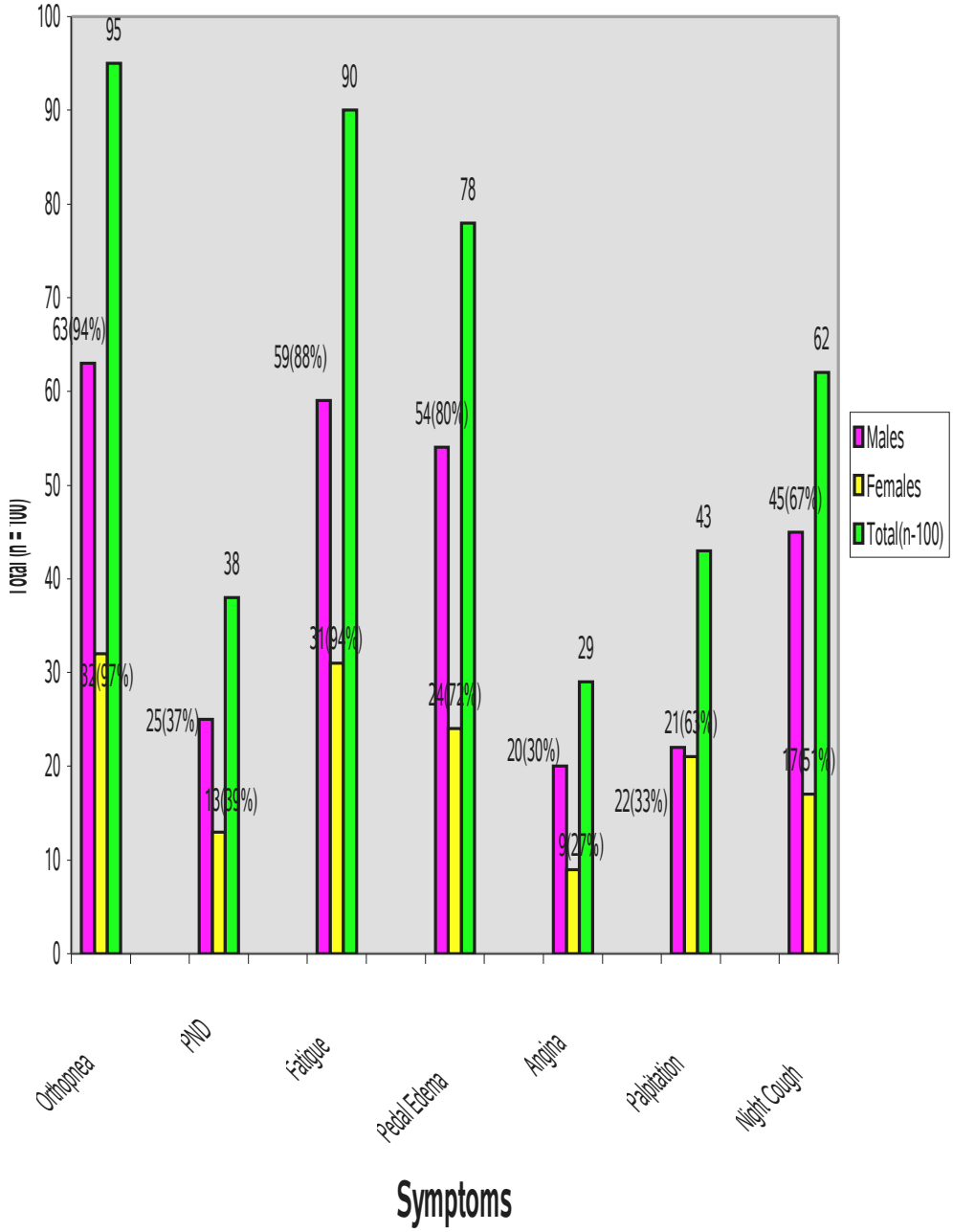


TABLE - 4

MAJOR RISK FACTORS

RISK FACTORS	Male	Female	Total
Diabetes	30	6	36
Hypertension	35	12	47
RHD	9	17	26
Cardiomyopathy	1	2	3
Others	0	0	0

Already existing structural heart disease is the major risk factor for CHF. 75% of patients had a history of heart disease in the past. Ischemic heart disease is the most common structural heart disease.

MAJOR RISK FACTORS

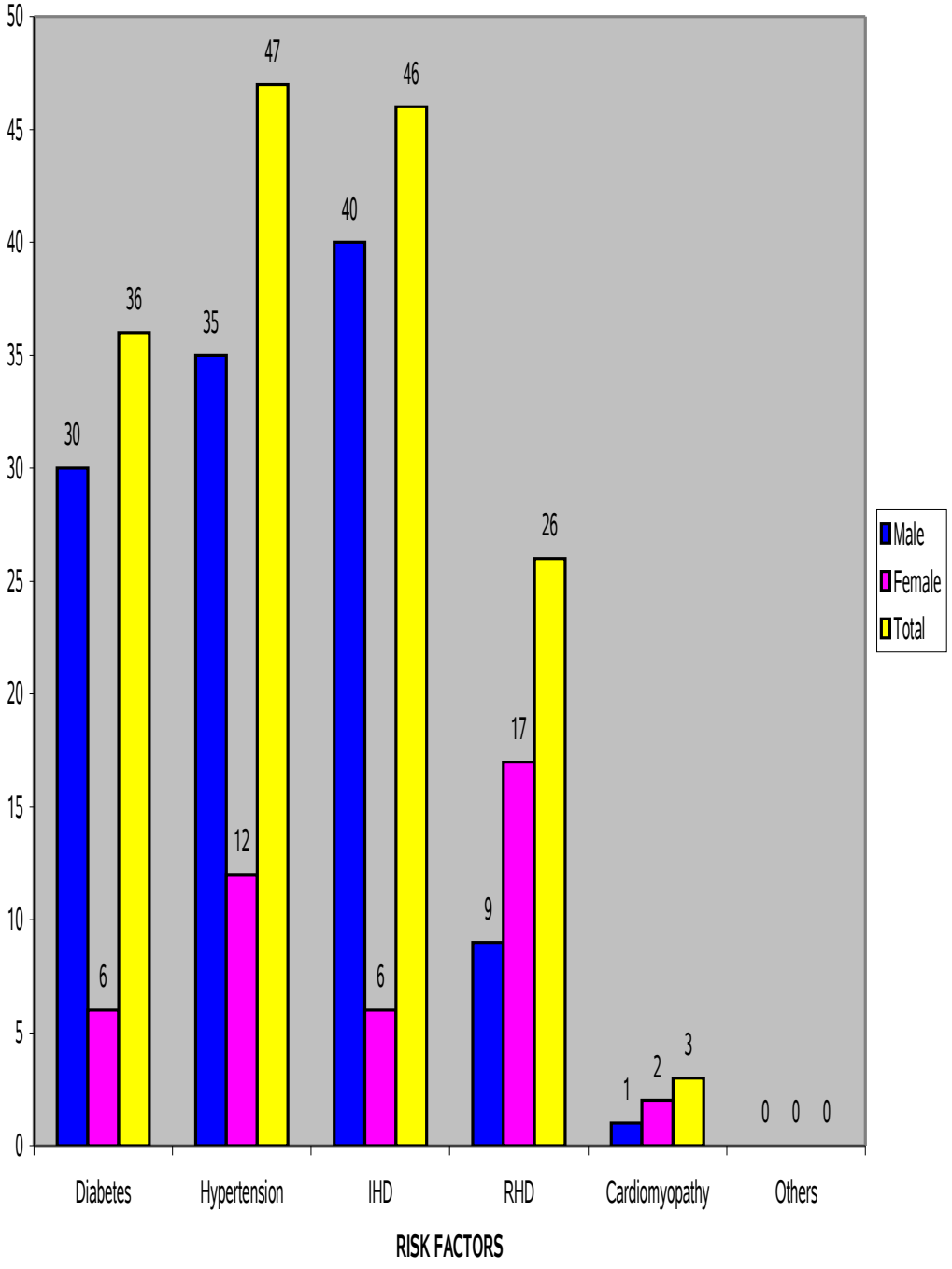


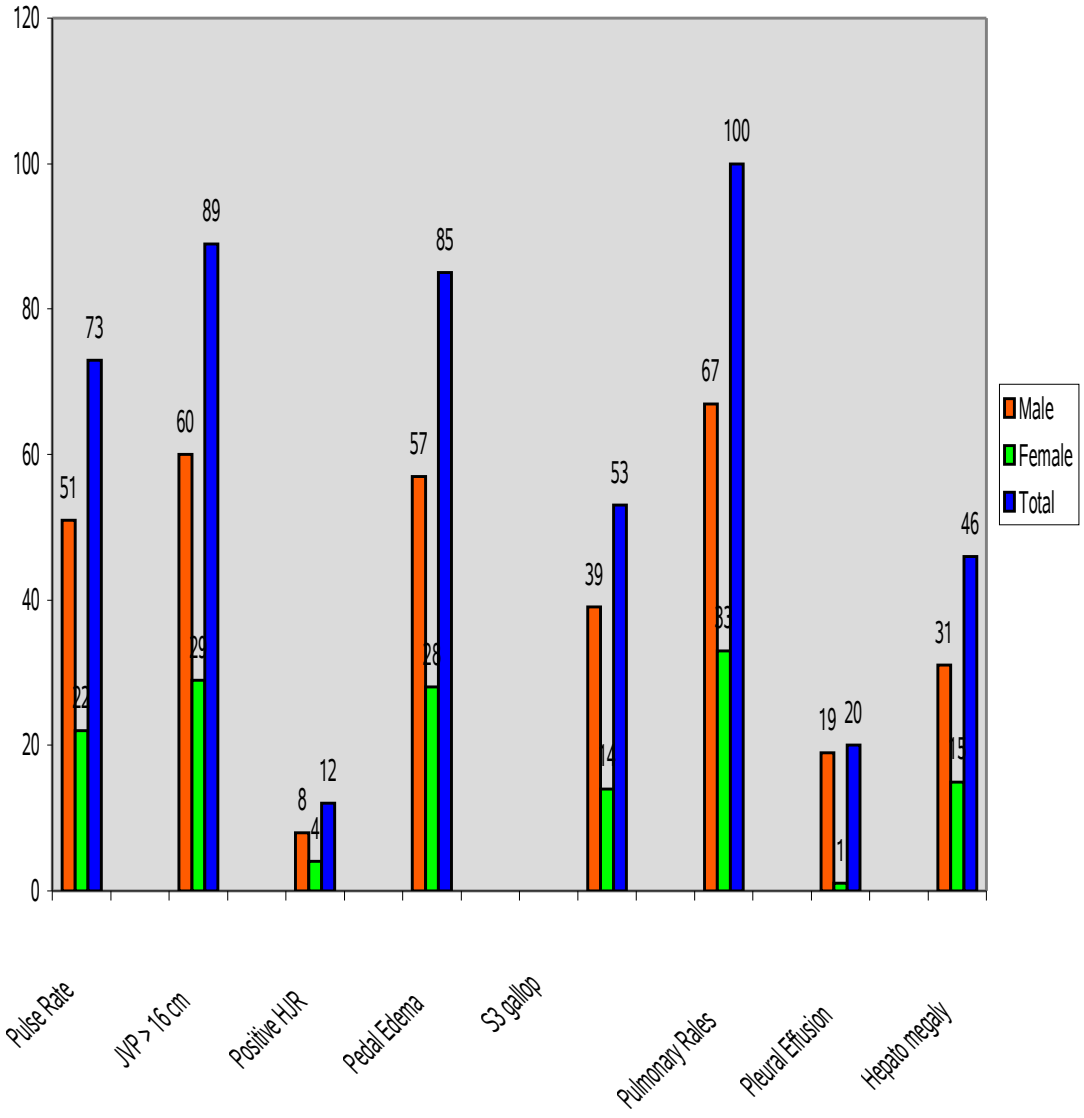
TABLE - 5

SIGNS

SIGNS	Male	Female	Total
Pulse Rate	51	22	73
JVP > 16 cm	60	29	89
Positive HJR	8	4	12
Pedal Edema	57	28	85
S_3 gallop	39	14	53
Pulmonary Rales	67	33	100
Pleural Effusion	19	1	20
Hepato megaly	31	15	46

The most common sign in CHF is Basal pulmonary rales followed by elevated JVP (89%) and pedal edema (85%)

SIGNS


120
100
80
60
40
20
0
51
22
73
60
29
89
8
4
12
57
28
85
39
14
53
67
33
100
19
1
20
31
15
46
Male
Female
Total
Pulse Rate
JVP > 16 cm
Positive HJR
Pedal Edema
S3 gallop
Pulmonary Rales
Pleural Effusion
Hepato megaly

SIGNS

TABLE - 6

CHEST X - ray

Sign	Total
Cardio megaly	91
Pleural effusion	17

Cardiomegaly is a sensitive sign of CHF occurring in 91% of patients.

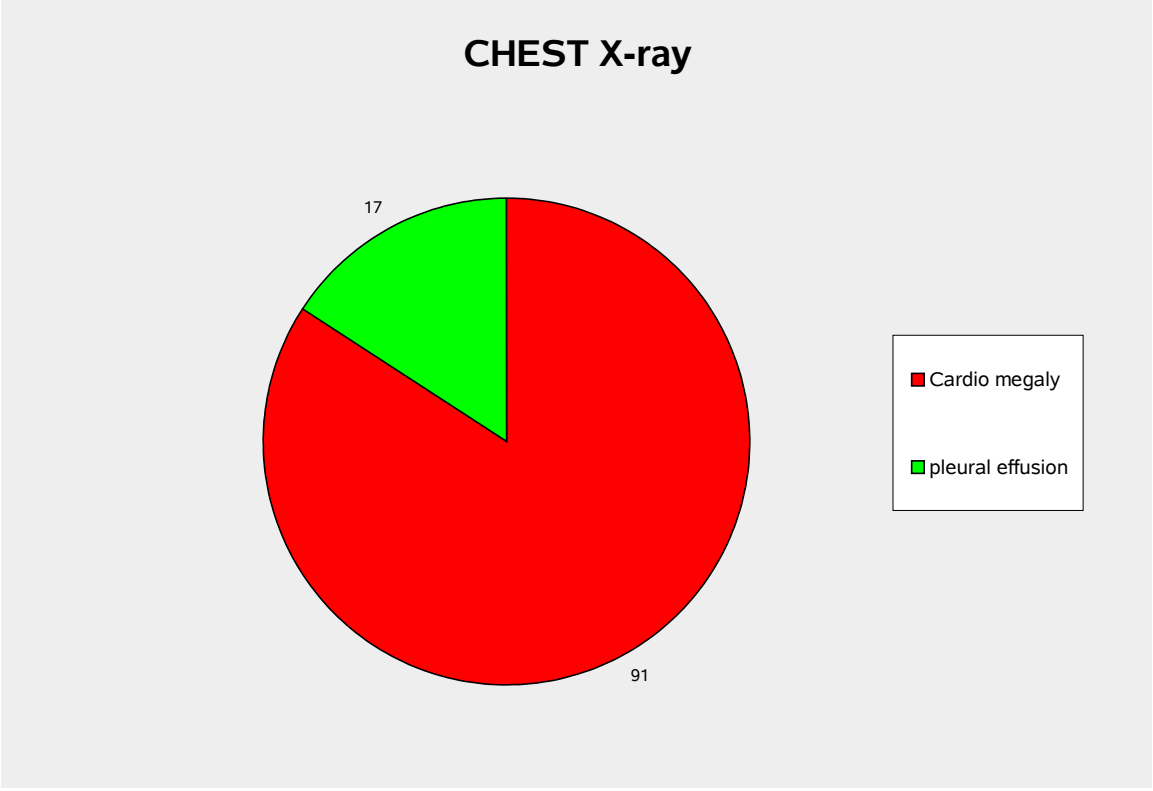


TABLE - 7

ECG

Sign	Total
Evidence of old MI	36
Ischemic changes	43
Atrial fibrillation	28
Left – Bundle branch block	17
Right Bundle branch block	15
Bifascicular block	9
Left ventricular hypertrophy	7

Electrocardiography evidence of IHD is found in 43% of patients. Atrial fibrillation is the only arrhythmia which had been documented apart from sinus tachycardia.

ECG

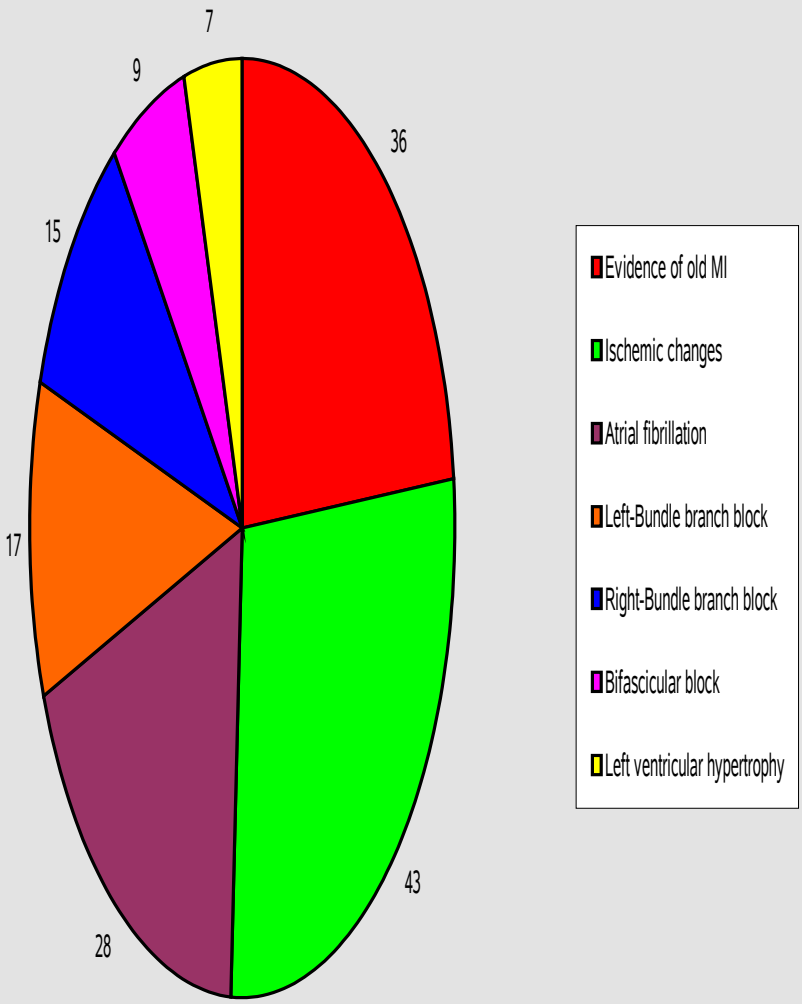

7
9
36
15
17
43
28
Evidence of old MI
Ischemic changes
Atrial fibrillation
Left-Bundle branch block
Right-Bundle branch block
Bifascicular block
Left ventricular hypertrophy

TABLE -7

ECHOCARDIOGRAPHIC FINDINGS

Ischemic Heart Disease

Sign	Total (n - 52)
Hypokinesia	37 (71%)
Akinesia	12 (23%)
Dyskinesia	3 (6%)
Ejection Fraction ≤ 40%	38 (73%)
Diastolic Dysfunction	13 (25%)

Depressed Ejection fraction is found in 73% of patients of IHD, while 25% of patients had Diastolic dysfunction.

ISCHEMIC HEART DISEASE

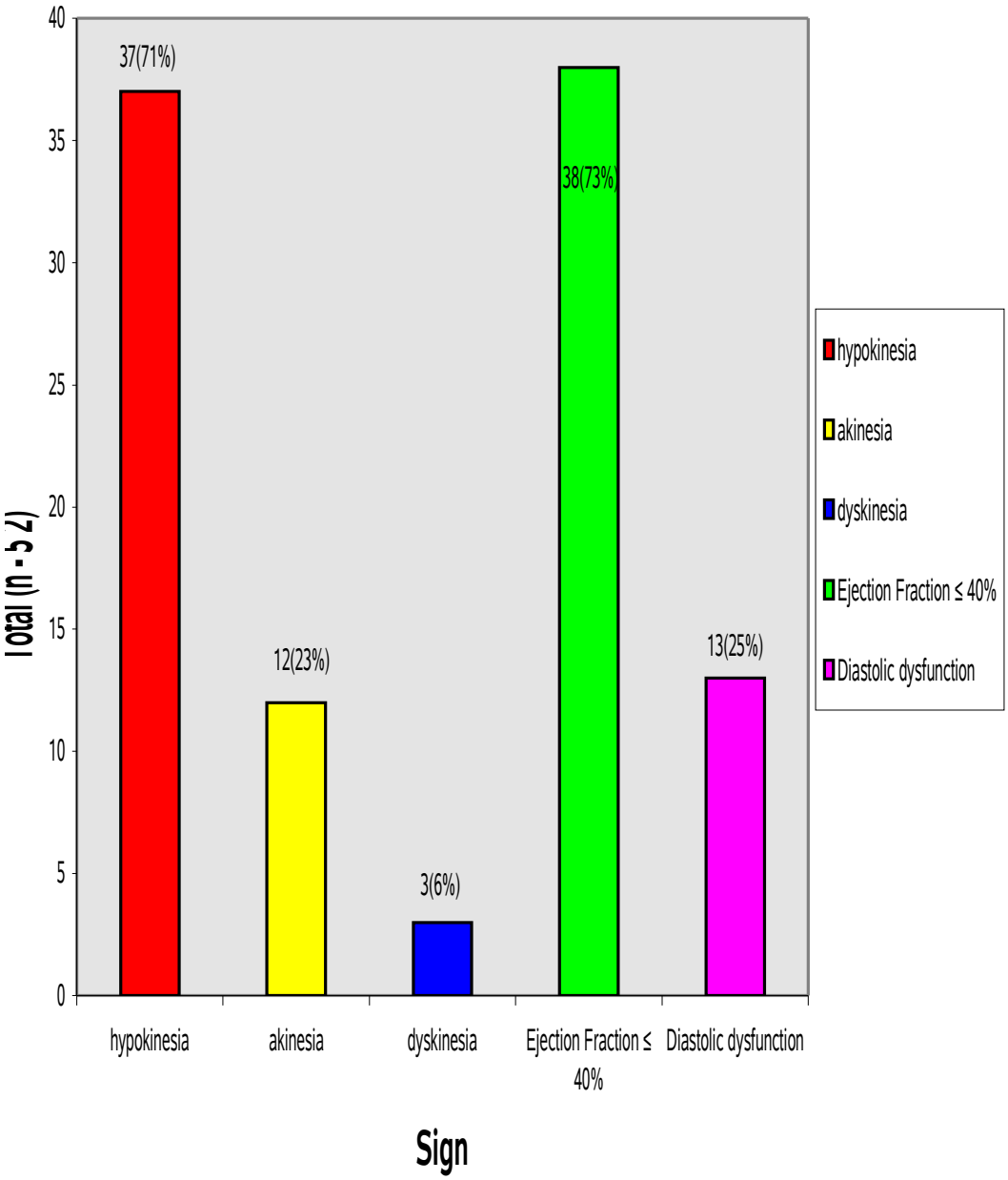


TABLE - 8

Rheumatic Heart Disease

Lesion	Total (n – 24)
Mitral Stenosis (MS) only	4 (16%)
Mitral regurgitation (MR) only	3 (12%)
Combined MS & MR	11 (46%)
Combined MS & Aortic regurgitation (AR)	2 (8%)
Combined AR + MR	4 (16%)

Among the Rheumatic Heart diseases, combined MS & MR accounted for a large proportion (46%) of cases of CHF.

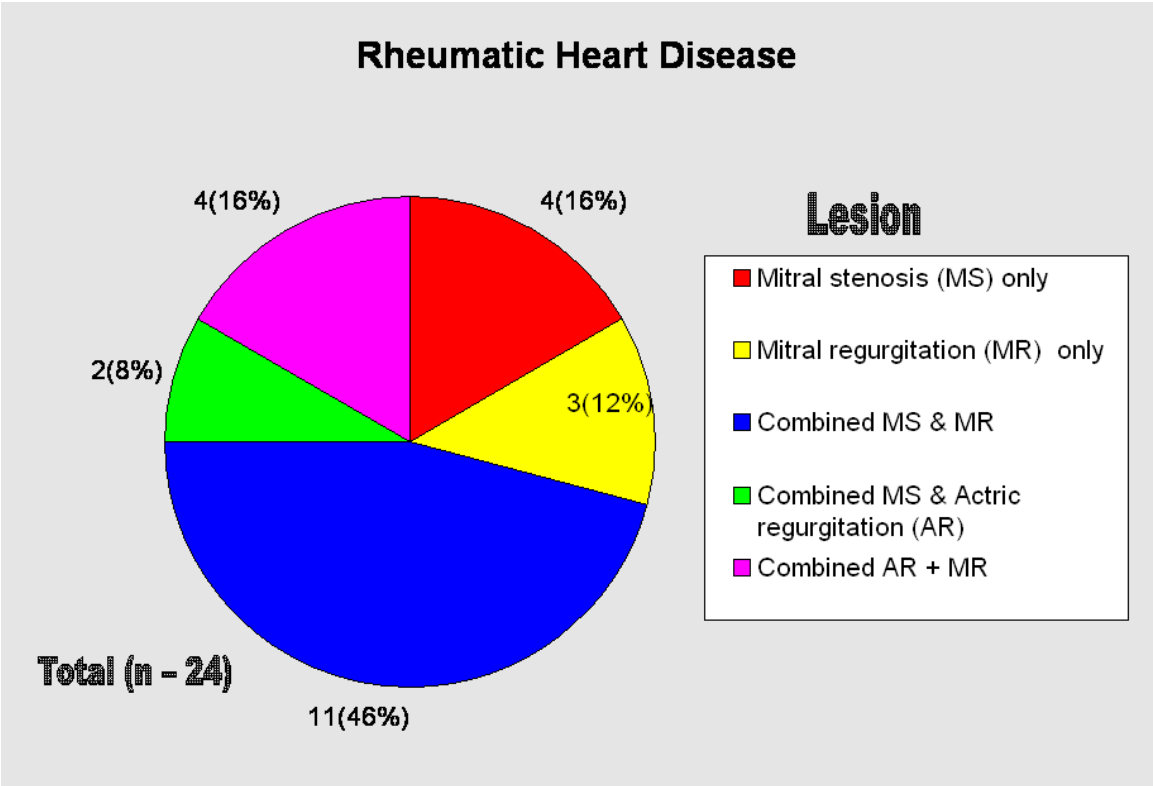

Rheumatic Heart Disease
4(16%)
4(16%)
2(8%)
3(12%)
Total (n – 24)
11(46%)
Lesion
Mitral stenosis (MS) only
Mitral regurgitation (MR) only
Combined MS & MR
Combined MS & Actric regurgitation (AR)
Combined AR + MR

TABLE – 9

Hypertensive Heart Disease

Sign	Total (n - 11)
Left Ventricular hypertrophy	11
Left atrial enlargement	2
Diastolic dysfunction	11

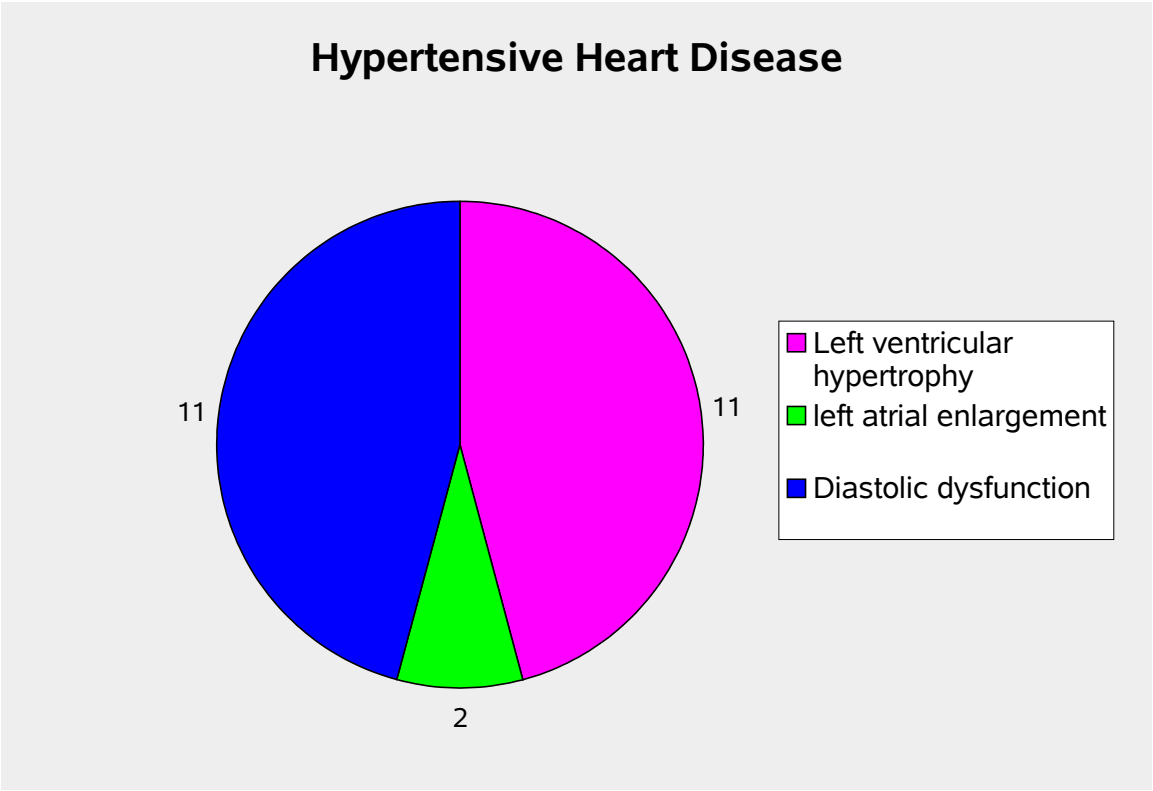


TABLE -10

Dilated Cardiomyopathy

Sign	Total (n – 9)
Ejection Fraction < 40 %	8 (88%)
Mitral regurgitation	4 (44%)

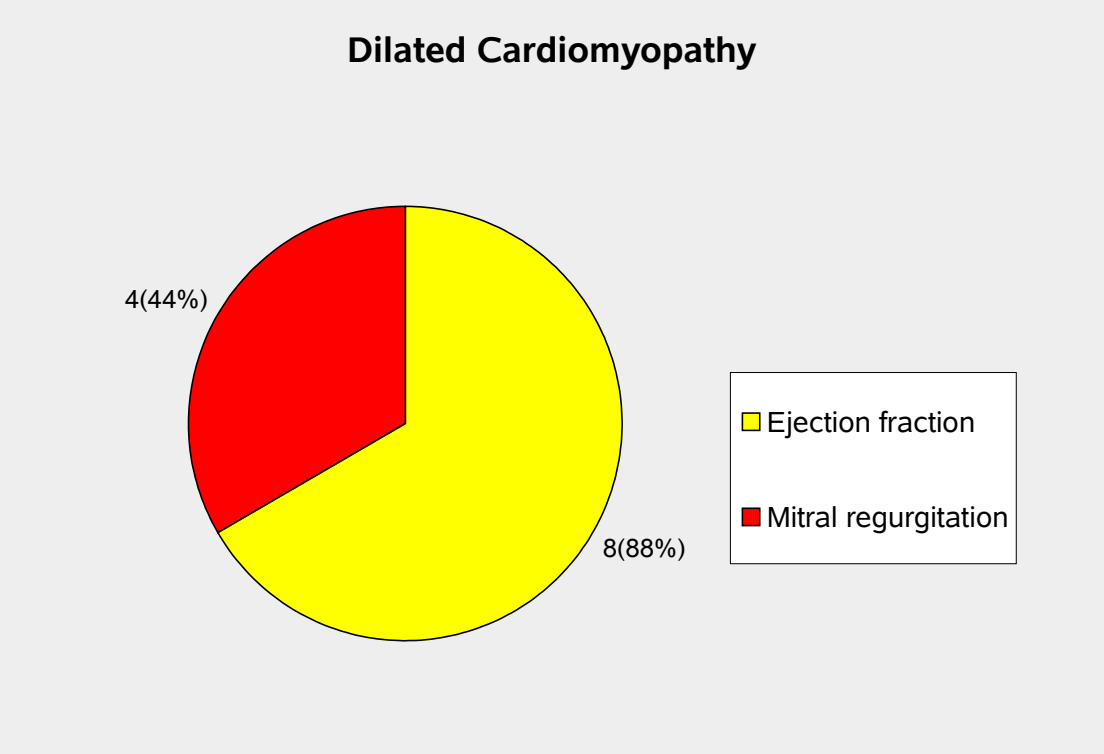


TABLE -11

ETIOLOGICAL PROFILE

Diagnosis	Male (n - 67)	Female (n - 33)	Total (n - 100)
Ischemic Heart disease	41 (61%)	11 (33%)	52
Hypertensive Heart disease	8 (12%)	3 (9%)	11

Rheumatic Heart disease	9 (13%)	15 (45%)	24
Dilated Cardiomyopathy	6 (9%)	3 (9%)	9
Anemia	3 (4%)	1 (3%)	4

Ischemic Heart disease accounts for a majority (52%) of cases of CHF. Rheumatic heart disease is the most common cause of Heart failure in females followed by Ischemic Heart disease.

ETIOLOGICAL PROFILE

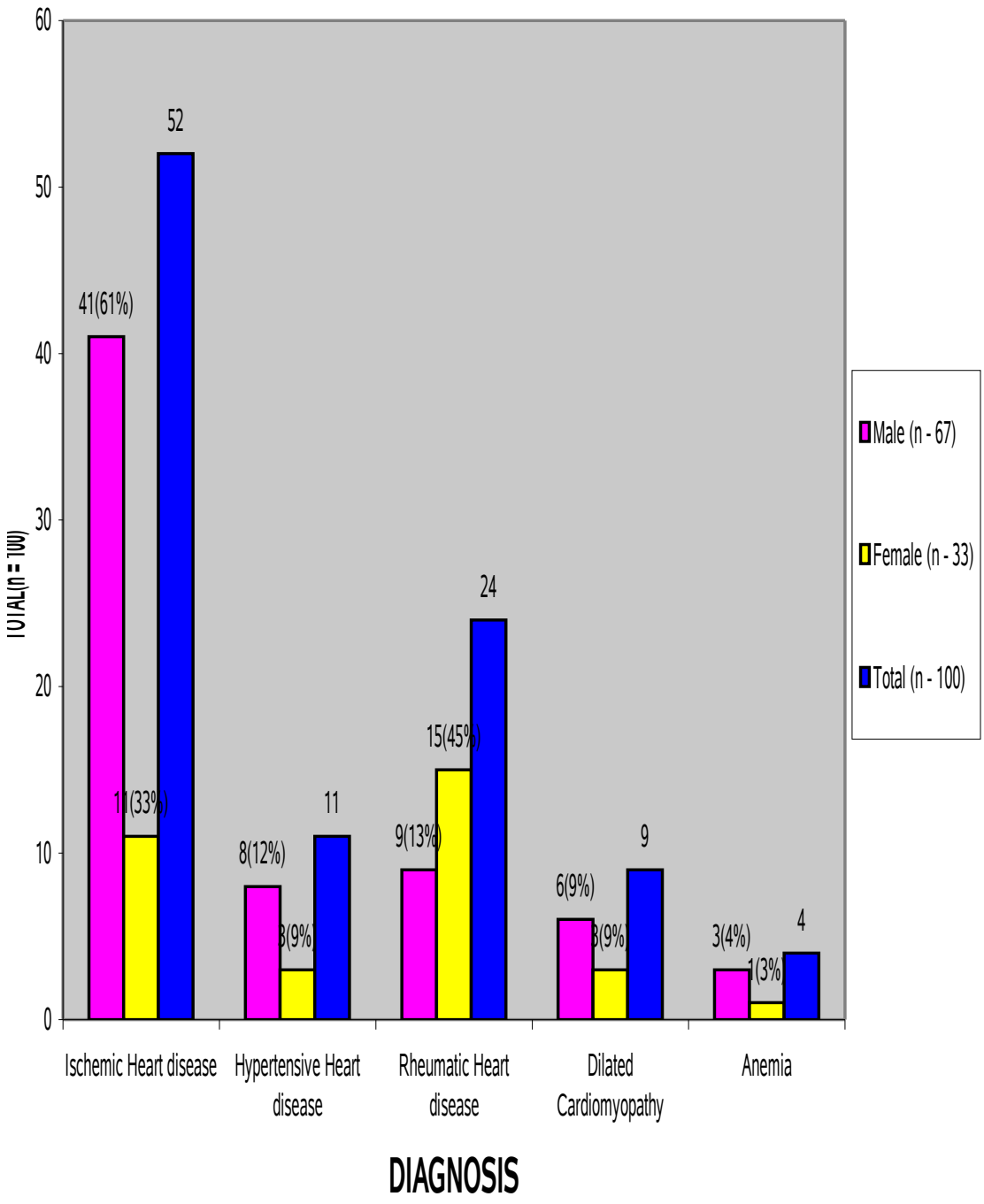
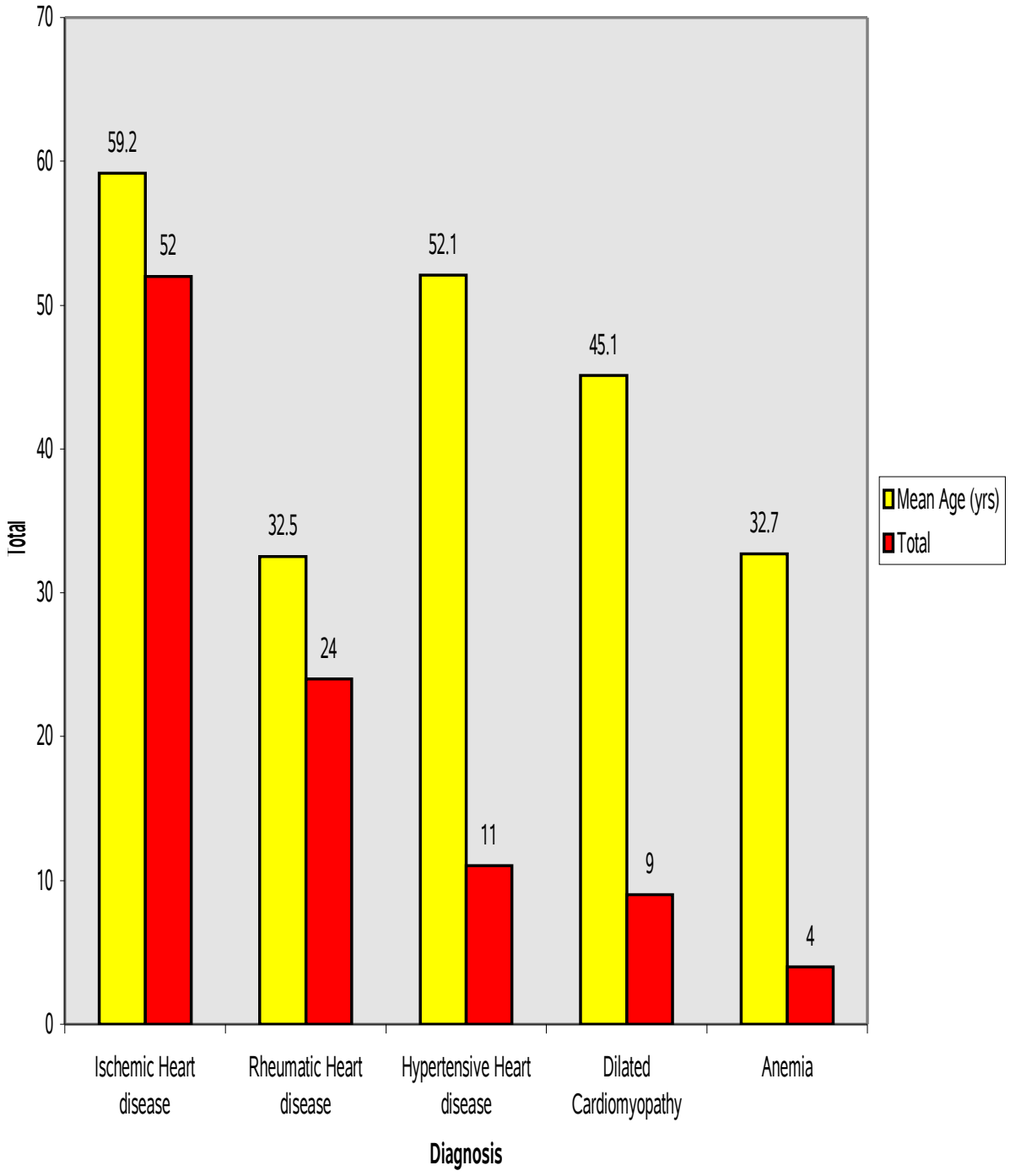


TABLE-12

Age and Heart Failure

Diagnosis	Mean Age (yrs)	Total
Ischemic Heart disease	59.2	52
Rheumatic Heart disease	32.5	24
Hypertensive Heart disease	52.1	11
Dilated Cardiomyopathy	45.1	9
Anemia	32.7	4

The mean Age of patients with CHF due to IHD was 59.2. The Mean age was low (32.5 yrs) for Rheumatic heart diseases compared to others.


Age and Heart Failure
70
60
50
40
30
20
10
0
Total
59.2
52
32.5
24
52.1
11
45.1
9
32.7
4
Ischemic Heart disease
Rheumatic Heart disease
Hypertensive Heart disease
Dilated Cardiomyopathy
Anemia
Diagnosis
Mean Age (yrs)
Total

DISCUSSION

The total number of patients analyzed were 100, out of which 67% were male patients. The Mean Age of patients found in the study was 50 years. Patients between the age group of 51 – 60 years accounted for a large proportion of cases.

The most common presenting complaint is dyspnea. Majority (59%) of patients with dyspnea were in class IV failure. This proportion is much higher compared to Euro heart failure survey where only 34% of patients had class IV failure.

Orthopnea is the second most common symptom constituting 95% of patients followed by Fatigue (90%) and Pedal Edema (78%).

RISK FACTORS:

Ischemic Heart disease is the most common structural heart disease predisposing to the development of chronic congestive Heart failure followed by hypertension and Diabetes. This finding correlate well with Framingham Heart study. Rheumatic heart disease accounts for a significant proportion

of heart failure in this study as opposed to the western data.

SIGNS:

Basal pulmonary rales is the most consistent sign associated with CHF followed by elevation of JVP (89%) and Pedal Edema (85%).

Chest skiagram revealed enlarged cardiac shadow in a majority of patients. As such chest X-ray can be considered as a sensitive test for the
initial diagnosis of CHF. Electro cardiographic evidence of Ischemic Heart disease is found in 43% of patients.

AETIOLOGY:

The most common cause of CHF in this study was Ischemic heart disease (52%) followed by Rheumatic heart disease (24%) and hypertensive heart disease. This study shows a higher prevalence of Rheumatic heart disease as a cause of CHF compared to western data. Combined valvular lesions are associated with increased risk of congestive heart failure.

ECHO CARDIOGRAPHY:

Among patients with non - valvular heart disease 61% of

patients had depressed ejection fraction (≤ 40%). Among patients with IHD 73% of patients had depressed ejection fraction. Diastolic dysfunction is present in 24 % of patients.

AGE AND CHF:

The Mean Age of patients with CHF due to IHD was 59.2 years whereas RHD leads to CHF at an earlier age (32.5 years). The presence of diastolic dysfunction in Hypertensive Heart disease leads to CHF at an earlier age (52.1 years) compared to Ischemic heart disease.

CONCLUSION

1. In this study 67% of patients were males and 33% of patients were females. The mean age of patients was 50 years.
2. The highest number of patients with CHF was in the age group of 51 – 60 years.
3. Ischemic heart disease was the most common risk factor for the development of chronic CHF.
4. Dyspnea was the most common presenting symptom and majority of them (59%) were in class IV failure.
5. Basal pulmonary rales was the most common clinical sign of CHF followed by elevated JVP and pedal edema.
6. Chest X-ray showed evidence of cardio megaly in majority (91%) of patients.
7. Atrial fibrillation was the only arrhythmia documented apart from sinus tachycardia.
8. The most common cause of congestive Heart failure is Ischemic heart disease (52%) followed by rheumatic Heart disease (24%) and Hypertension (11%).

9. Among the patients with non-valvular heart disease, 61% had depressed ejection fraction (≤ 40%) while 24 % had Diastolic dysfunction
10. Rheumatic heart disease accounted for a majority of cases of CHF, relatively at an earlier age (Mean 32.5 years) compared to IHD (Mean 59.2 years).

BIBLIOGRAPHY

1. Anderson RN: United States Life tables, 1998: National Vital Statistics Reports. In Hyaltsville, (Eds) National centre for healthy Statistics, 2001: vol 48, no, 18.
2. American diabetics Association: Clinical practice recommendation 2002. Diabetes Care 2004, 27: 51.
3. Bourassa MG, Gurae O, Bangdiwala S,et al. Natural History and patterns of current practice in heart failure. J. Am. Coll Cardiol, 1993,22: 14 – 19.
4. Braunwald, E. Report of the task force on Research in heart failure, Bethesa M.D., National Heart, Lung and Blood Institute, 1994.
5. Cowie MR, Wood DA, Coats AJS, Thompson SG, Poole Wilson PA,Suresh V, et al. Incidence and aetiology of heart failure: a population- based study. Eur heart J 1999; 20: 421 – 8.
6. The CONSENSUS Trail study group, Effect or enalapiril on morality in severe congestive heart failure: results of the cooperative north Scandinavian enalapiril survival study (CONSENSUS). N Engl. J Med. 1987: 1429-35.
7. Chobanian Av, Bakris GL, Black HR, et al. Seventh report of the joint National committee on the prevention, Detection, Evaluation and treatment of High blood pressure. Hypertension 2003: 42: 1206 – 1252.
8. Colucci WS et al. Clinical aspects of heart failure, in Heart disease, 6th ed, E

Braunwald (ed), Philadelphia Sounders.

9. Davis R.C. Hobbs, FDR Lip. GH History and Epidemiology. ABC of Heart failure: BMU: 2003: 1-4.

10. Dallus, Tx American heart association 2000, Heart and stroke statistical update, 2000: vol 3, 56-68.

11. Dallas Volte S, A short historical survey of heart failure. J. Chin Invest 1993: 71: S167 – S176.

12. Esler M, Kaye D, Lambert G, et, al. Adrenergic nervous system in heart failure, Am J Cardiol 1997, 80: 71 – 141.

13. Francis GS, Benedict C, John stone De, et al. Comparison of Neuro endocrine activation in patients with left ventricular dysfunction with and without congestive heart failure. A sub study of the studies of LV dysfunction. (SOLVD) Circulation 1990, 82: 1724 – 1729.

14. Feildman MD, Alderman JD, Aroesty JM et al. Depression of systolic and diastolic myocardial reserve during artial tachycardia in patients with dilated cardiomyopathy. J Chin Invest 1998, 82: 1661.

15. Gheorghiade M, Bonow RO. Chronic heart failure in the United States : A manifestation of coronary artery disease circulation 1998, 97 : 282-289.

16. Grantham JA, Borgeon DD, Burnet et al. Pathophysiological and potential therapeutic roles in CCF. Am J Physiol 1997, 92: 12, 1077-1083.

17. Goldsmith SR, Francis GS, Cowley Aw. Arginine Vasopressin and the renal

response to water loading in CHF. Am J Cardiol 1986, 58: 295.

18. Gaash WH. Diagnosis and treatment of heart failure based on left ventricular function Am J of cardiology 1999,65:23

19. Ghali JK Kadakin S. Cooper R, Farlinz precipitating factors leading to decomposition of heart failure: Trails among urban blacks. Arch Intern Med. 1988, 148: 2013.

5) Goldman L, Hasimto B, Cook EF, Loscalzo A. Comparitive reproducibility and validity of symptoms for assessing cardiovascular functional class: Advantages of a new specific activity scale. Circulation 1981, 64: 1227.

6) Horine Ef. An epitome of ancient pulse core: Bull Hist, Med, 1941: 10: 209-249.

7) Ho KKL, Pinksy JL, Kannel WB, et al, The epidemiology of heart failure : The framingham study. J Am Col Cardiol 1993, 22: 6A – 13A.

8) Haldeman GA, Croft JB, Giles WH. Rashidee A. Hospitalisation of patients with heart failure: National Hospital discharge survey. 1985 10 1995. Am Heart J.1999, 137: 352.

9) Ibrahim BS. The frequency of systolic versus diastolic heart failure in an Egyptian cohort. Eur J Heart fail 2003, 5: 41-5.

10) International Agency for Research on Cancer: Tobacco smoke and Involuntary smoking, lARC Monographs on the Evaluation of Carcinogenic Risks to Humans. Lyon, France, 2003, Vol 83.

11) Khadra A, Salem DN, Rand W. Antiplatelet agents, and survival: A coherent

analysis from the study of Left Ventricular dysfunction (SOLVD) Trial Jam Coll Cardiol, 1988, 31: 419 – 425.

12) Kitzman DW, Little WC, Brubaker PH, Anderson RD, Hindley WG, Manberger Ct et al, Pathophysiological characterization of Isolated diastolic heart failure in comparison to systolic heart failure. Ia MA 2002: 288: 2144 – 50.

13) Lip GYH, Sarwar S, Ahmed J, Lee S, Kapoor V, Child D, et al, A survey of heart failure in general practice the West Birmingham heart failure project. Eur J Gen Prac. 1997: 3 : 85-9.

14) Mancini DM, Wilson JR, Bolinger L, et al. Invivo magnetic resonance spectroscopy measurement of deoxymyoglobin during exercise in patients with heart failure. Demonstration of abnormal muscle metabolism despite adequate oxygenation Circulation, 1994, 90: 500.

15) Mannin HL, Schwartzateiu RM. Mechanisms of disease: Pathophysiology of dyspnoea: N Eng J Med, 1995 333: 1547.

16) Milne EN, Pistolesi M, Miniati M, Giuntiwi C: The radiologic distinction between cardiogenic and non cardiogenic pulmonary oedema. ASR Am J Roentgenol 1985, 144: 879.

17) Massie BM, Shah NB: Evolving trends in the epidemiologic factors of heart failure: Rationale for preventive strategies and comprehensive disease Management, Am Heart J 1997, 133: 703.

18) Maki T, Toinoven, Koshinen P, et al. Effect of ethanol drinking, hangover and

exercise on Adrenergic activity and heart rate validity in patients with a history of alcohol induced AF. Am J Cardiol, 1998, 82: 317.

19) Moe GW, Grima EA, Wong NL, et al. Dual natriuretic peptide system in experimental heart failure, J Am Coll Cadiol 1993, 22: 891.

20) Moore DA, William withering and Digitalis BMJ, 1985: 290 – 324.

21) Oakley C, Biology, Diagnosis, investigations and management of cardiomyopathies. BMJ 1992: 315: 1520 – 4.

22) Parker M, Survival in patients with chronic heart failure and its potential modification by drug therapy. In Cohn JN (ed Drug treatment of heart failure, 2nd ed. Seeannus NJ, ATC international, 1988, p273.

23) RKG MW, Nease RF. Cost effective analysis in clinical practice: A case of heart failure, Arch internal Med 1999, 159: 1690.

24) Richs. Pulmonary hypertension and Corpulmonale. In Braunwalds Heart diseases 7th edition. Zippe D, et al, Saunders publications philadephia, USA, 2005, 1113-1115.

25) Rahimtoola Sh. The hibernating myocardium. Am Heart J 1989, 117: 2111-21.

26) Senni M, Triboulloy CM, Rodeheffer RJ, et al. Congestive heart failure in the community. Arch Inten Med, 1999, 157: 29-34.

27) Stevenson WG. Mechanism and management of arrhythmias in heart failure. Curv, Opin Cardiol, 1994, 10: 274.

28) Schoffer J, Tews A, Langes K et al. Relationship between myocardial nor epinephrine content and left ventricular function and endomyocardial biopsy study. Eur Heart J 1987, 8 : 748.

29) Stevenson LW, Perloff SK. The limited reliability of physical signs for estimating hemodynamics in chronic heart failure JAMA 1989, 261: 884.

30) The task force on Heart failure of the European Society of Cardiology, Guidelines for the diagnosis of heart failure. Eur Hear J 1995: 6: 741 – 51.

31) JN. Goldhaber SZ, P, Pfeffer in A, An overview of contemporary etiologies of CH. Am Heart J 1991; 121: 1852.

32) Tsutamoto T, Hisanaga T, Fukai D, et al. Prognostic value of plasma intercellular, adhesion molecule and endothelin l, concentration in patients with chronic congestive heart failure. Am J Cardiol, 1995 76: 803 – 808.

33) Van Cheng BS, Kazanagra K, Garcia A, et al. A rapid bedside test for B type natriuretic peptide treatment outcomes in patients admitted with decompensated heart failure. J Am Coll. Cardiol, 2001, 37 : 386 – 391.

34) Vasan RS, Levy D. Defining diastolic heart failure: a call for standardized diagnostic criteria. Circulation 2000, 101: 2118 – 21.

35) Vasan RS, Benjamin EJ, Levy D, Prevalence, clinical features and prognosis of. diastolic heart failure: an epidemiologic perspective. J Am Coll Cardiol, 1995, 26 : 1565

36) Yanagisawa M, Kurihara H, Kimura s et al. A novel potent vasoconstrictor

peptide produced by vascular endothelial cells, Nature 1988, 332: 411.

37) Yusuf S, Sleight, Pogne J et al. Effects of an angiotensin converting enzyme inhibitor, ramipril on cardio vascular events in high risk patients: the outcome prevention evaluation study. N Engl J Med, 2000, 342:145.

38) Zuccala G, Galtel C, Manas Grarina E, et al. Left Ventricular dysfunction. A clue to cognitive impairment in older patients with heart failure. J Neurol Neurosurg. Psychiatry 1997, 63: 509.

PROFORMA

HEART FAILURE

Name : I.P.NO :

Age : Sex :

Occupation : Income :

Residence :

Present Illness :

1.Breathlessness Yes / No

Class I II III IV

2.Orthopnea Yes / No

3.PND Yes / No

4.Night Cough Yes / No

5.Chest Pain Yes / No

6.Palpitation Yes / No

7.Syncope Yes / No

8.Pedal Edema Yes / No

9.Oliguria Yes / No

10.Nocturia Yes / No

Past History :

1.Diabetes Yes / No

2.Hypertension Yes / No

3.IHD Yes / No

4.Cardiomyopathy Yes / No

5.Congenital Heart Disease Yes / No

6.RHD Yes / No

7.Thyroid Disorders Yes / No

8.Anemia Yes / No

9.CKD Yes / No

10.Others Yes / No

Personal History

1.Smoking Yes / No

2.Alcoholism Yes / No

3.Drug Abuse Yes / No

4.STI Yes / No

Family History

1.Diabetes Yes / No

2.Hypertension Yes / No

3.Heart Disease Yes / No

Examination :

1.Consciousness Normal / Altered

2.Pallor Yes / No

3.Dyspnea Yes / No

3.Cyanosis Yes / No

4.Clubbing Yes / No

5.Extremity Edema Yes / No

6.JVP Raised / Not Raised

7.Blood Pressure : Temperature : Weight :
8.Pulse :

9.Respiratory Rate :

Cardio-Vascular System :

1.Heart Sounds

2.Murmur

3.S 3

Respiratory System:

1.Rales Yes / No

2.Pleural Effusion Yes / No

Abdomen:

1.Hepatomegaly Yes / No

CNS :

Investigations :

1.Hemogram:

Hb

Tc

Dc

ESR

2. Blood:

Sugar

Urea

Creatinine

Na K Cl Hco3

3.Chest X-Ray:

4.ECG :

5.Echocardiography:

Final Diagnosis :

ABBREVIATIONS

ACE – Angiotensin Converting Enzyme

ANM-anemia

AS – Aortis Stenosis

AR – Aortic Regurgitation

BP –Blood Pressure

CHF – Congestive Heart Failure

CM – Cardiomegaly

DC – differential count

CXR – Chest X-Ray

DM – Diabetes Mellitus

DCMP – Dilated Cardiomyopathy

ESR – erythrocyte sedimentation rate

EF – Ejection Fraction

Hb – Hemoglobin

HT – Hypertension

HJR – Hepatojugular Reflex

HHD – Hypertensive Heart Disease

IHD – Ischemic Heart Disease

JVP – Jugular Venous Pressure

LV – Left Ventricle

MS – Mitral Stenosis

MR – Mitral Regurgitation

PR – Pulse Rate

PF – Pleural Effusion

RHD – Rheumatic Heart Disease

RV – Right Ventricle

TC – total white blood cell count

MASTER CHART

1 – Yes

2 - No